Manual Therapy xxx (2013) 1–5

Contents lists available at ScienceDirect

Manual Therapy

journal homepage: www.elsevier.com/math

Review article

The central nervous system – An additional consideration in 'rotator cuff tendinopathy' and a potential basis for understanding response to loaded therapeutic exercise

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ARTICLE INFO

Article history:
Received 6 February 2013
Received in revised form
2 July 2013
Accepted 15 July 2013

Keywords:
Rotator cuff tendinopathy
Exercise
Rehabilitation
Pain

ABSTRACT

Tendinopathy is a term used to describe a painful tendon disorder but despite being a well-recognised clinical presentation, a definitive understanding of the pathoaetiology of rotator cuff tendinopathy remains elusive. Current explanatory models, which relate to peripherally driven nocioceptive mechanisms secondary to structural abnormality, or failed healing, appear inadequate on their own in the context of current literature. In light of these limitations this paper presents an extension to current models that incorporates the integral role of the central nervous system in the pain experience. The role of the central nervous system (CNS) is described and justified along with a potential rationale to explain the favourable response to loaded therapeutic exercises demonstrated by previous studies. This additional consideration has the potential to offer a useful way to explain pain to patients, for clinicians to prescribe appropriate therapeutic management strategies and for researchers to advance knowledge in relation to this clinically challenging problem.

1. Introduction

Tendinopathy is a term commonly used to describe tendon pathology and/or pain. Despite being a well-recognised clinical presentation, a definitive understanding of the pathoaetiology of rotator cuff tendinopathy remains elusive (Lewis, 2009). Over recent years there has been a focus upon understanding pain associated with tendinopathy from the perspective of local tissue based pathology. But, in light of the well-recognised dissociation between pathology and pain (Cook and Purdam, 2009; Drew et al., 2012), it is becoming clear that additional explanatory models are now needed (Drew et al., 2012).

In view of this, the aim of this paper is to present a theoretical extension to current models incorporating the integral role of the central nervous system (CNS) in the pain experience. For the purpose of clarity within this paper and to aid clinical translation, the terminology 'rotator cuff tendinopathy' refers to a presentation where a person complains of shoulder pain with movement that is provoked further with load, for example lifting or through resisted tests performed by a clinician during a physical examination (Littlewood et al., 2012a).

We recognise that the reader might object to or question the appropriateness of the term rotator cuff tendinopathy for two reasons. Firstly, the criteria we use to define rotator cuff tendinopathy is broad and might include a range of biomedical diagnoses, including subacromial impingement, subacromial bursitis, rotator cuff tear, acromioclavicular joint osteoarthritis etc. However, in the absence of evidence to support the validity or reliability of such diagnoses (May et al., 2010), particularly in relation to the lack of association between pathology and pain, it is difficult to substantiate such an objection. Secondly, in the context of attempts to highlight the role of the CNS, such specific pathology or impairment terminology might be regarded as a backwards step because of their reference to specific peripheral tissue or mechanical mechanisms. However, such a broad definition of tendinopathy in this

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http://dx.doi.org/10.1016/j.math.2013.07.005

translational paper is deliberate and purposeful to highlight how current practice models can be interpreted and usefully enhanced without wholesale, probably unrealistic, changes to practice and terminology; hence there is pragmatic value.

A secondary aim is to offer a potential rationale to explain the favourable response to loaded therapeutic exercises demonstrated by previous studies (Jonsson et al., 2005; Bernhardsson et al., 2010; Holmgren et al., 2012; Littlewood et al., 2012a). These further considerations have the potential to offer a useful basis upon which to explain pain to patients and for clinicians to prescribe appropriate therapeutic management strategies.

2. Local tissue pathology-pain models

This paper will begin by offering a critique of local pain models as a basis upon which to justify the need for greater consideration of the CNS. Tissue based pathology-pain models have been proposed (Cook and Purdam, 2009) and adapted to the rotator cuff (Lewis, 2010). However, as mentioned, these models are confounded by the lack of association between pathology and pain (Cook and Purdam, 2009; Drew et al., 2012). Using magnetic resonance imaging, Frost et al. (1999) could not distinguish individuals diagnosed with subacromial impingement from asymptomatic age-matched controls according to structural pathology. In keeping with this, up to 40% of the general population have asymptomatic rotator cuff tears (Templehof et al., 1999; Worland et al., 2003; Yamamoto et al., 2010). Studies investigating prognosis (van der Windt et al., 1996; Bonde et al., 2003; Ekeberg et al., 2010) have suggested that the biomedical diagnosis, relating to specific tissues at fault, was not associated with clinical outcomes. Furthermore, it has been reported that structural change does not explain response to therapeutic exercise because as clinical outcomes improve a corresponding change in observable structural pathology is not seen (Drew et al., 2012). Hence, in the context of this literature, traditional models that describe tissue injury/structural pathology resulting in nocioceptive input and a pain response in proportion to the extent of injury seem inadequate, if considered in isolation.

3. Local biochemical models

In light of the shortcomings of local tissue pathology-pain models, others have suggested a local biochemical basis for the pain associated with tendinopathy where biochemical mediators in the tissue stimulate nocioceptive afferent fibres (Khan et al., 2000). Degenerative pathology is associated with neurovascular ingrowth and potential pain mediators such as substance P and acetylcholine. However, it remains unclear whether biochemical substances are a cause of tissue degradation and/or pain or whether they are a by-product of tendinopathy (Danielson, 2009). But, because biochemical models make no assumption about the underlying pathology, such biochemically driven nocioceptive pathways might offer further understanding of symptomatic versus asymptomatic pathology. Further research in this area is on-going (Rees et al., 2013).

So, in light of what is currently known, local biochemical models appear to have the potential to enhance understanding and management of tendinopathy. But, neither these or local tissue pathology-pain models recognise the role of the CNS nor critically that nocioception is neither sufficient nor necessary for a pain experience (Moseley, 2007).

4. Background to the role of the CNS

A contemporary understanding of pain suggests that there might be other mechanisms involved in pain associated with tendinopathy that might act with the local mechanisms outlined above or in isolation. The notion that the state of the tissue does not provide an adequate measure of pain is recognised in relation to other pain syndromes (Moseley, 2007; Melzack and Wall, 2008) but in tendinopathy local tissue/biochemical based models are predominantly used to explain pain (Cook and Purdam, 2009; Lewis, 2010; Liu et al., 2011). Such models continue to be developed but fail to adequately recognise the integral role of the CNS in the pain experience. This omission neglects a whole body of pertinent literature, that might offer some further explanation as to why attempts to link symptoms to peripheral structural pathology continue to fall short (Moseley, 2007; Wand et al., 2011).

We suggest here that the pain associated with rotator cuff tendinopathy, that persists beyond expected recovery times, should be evaluated within a framework that recognises the potential for altered processing and modulated output of the CNS rather than solely a product of peripherally driven nocioception secondary to persistent tissue abnormality, for example tendon degeneration or tear. Note that we have used the term recovery time as opposed to healing time because many studies suggest that the rotator cuff does not always 'heal' from a structural perspective, even after attempts to surgically repair torn tissue (Galatz et al., 2004; Rees et al., 2006) although symptoms might still improve over time. In this context it is difficult to define a definitive time point by which we can assert that peripheral tissue recovery has been completed in terms of the inflammatory and proliferative stages. It is likely that this point will be highly individualised and compounded by factors specific to the rotator cuff including the relative hypovascularity of the tissue (Rees et al., 2006; Lewis, 2010). In practice, it might be more important to consider factors other than time-course of symptoms when considering whether local or CNS pain mechanisms predominate.

5. Explaining pain

The following section describes the potential mechanisms involved in pain associated with rotator cuff tendinopathy. The aim is to offer a reasoned explanation as to why pain state or output might persist and might not be proportionate to the state of the rotator cuff tissue. In addition to enhancing understanding of pain mechanisms, one further consequence of this might be a direct challenge to current practice where, for example, prescription of loaded exercise is limited due to fear of causing tissue damage (Littlewood et al., 2012b).

5.1. Central mechanisms

We begin by considering potential aberrations relating to processing of afferent inputs at the spinal cord level. Central sensitisation is a state that has been described in terms of altered processing where dorsal horn cells in the spinal cord become increasingly sensitised (Gifford, 1998a). In this altered state even non-noxious input, for example lifting the arm, can contribute to a painful output (Gifford, 1998a). Gwilym et al. (2011), recognising that anomalies existed between peripheral tissue structure and the degree of pain experienced, proposed the presence of central sensitisation in a significant proportion of their patients who underwent subacromial decompression. Furthermore, those patients who were regarded as having greater levels of central sensitisation pre-operatively reported worse outcomes three months following the operation. Clearly, pain mechanisms beyond peripherally driven nocioceptive mechanisms are in play here and the study by Gwilym et al. (2011) casts further doubt upon the validity of tissue state as the sole basis upon which to understand pain.

Please cite this article in press as: Littlewood C, et al., The central nervous system – An additional consideration in 'rotator cuff tendinopathy' and a potential basis for understanding response to loaded therapeutic exercise, Manual Therapy (2013), http://dx.doi.org/10.1016/j.math.2013.07.005